Operative Techniques in
Thoracic and
Cardiovascular Surgery

Endovascular Therapy for Malperfusion in Acute Type B Aortic Dissection

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Presentation with acute type B aortic dissection (B-AD) in the setting of malperfusion is associated with a high risk for major morbidity due to end-organ ischemia. This presentation has traditionally been considered an indication for operation where the entry tear is resected, and branch vessel perfusion is subsequently restored either as a result of the aortic repair or by direct revascularization of the peripheral arterial bed.

There are two predominant mechanisms by which malperfusion can occur, and these have previously been defined by our group.[1,2] In static obstruction, the dissection flap enters the branch vessel lumen without an adequate reentry tear (or a diminutive reentry tear) within the course of that artery. The compromised true lumen of that artery then becomes the sole source of inflow into that end organ. In contrast, in dynamic obstruction, the mobile aortic dissection flap intermittently covers the orifice of the branch vessel during the cardiac cycle, thus impeding arterial inflow into the end organ. Treatment is directed at restoring the integrity of the arterial true lumen in static obstruction and of the aortic true lumen in dynamic obstruction. Finally, end-organ perfusion may be compromised by a combination of both mechanisms, as well as by miscellaneous causes including thrombosis or embolism of branches supplied by either lumen.

Whereas malperfusion has traditionally been an indication to proceed with early operative repair of the aorta, endovascular approaches have emerged as an alternative to allow restoration of end-organ perfusion. Two types are discussed in this report, and each has a different focus. The traditional endovascular approach has relied on a percutaneous method of creating a fenestration in the dissection flap to equalize pressures and allow flow between the true and false lumina. A self-expanding stent (typically 16 to 22 mm in diameter) is then placed in the aortic true lumen to maintain its patency and thereby allow continued antegrade perfusion of the branch vessel. This approach typically resolves the dynamic type of obstruction. If static obstruction is identified, self-expanding stents are placed within the true lumen of the branch vessel, much in the same way typical obstructive arterial lesions are treated. The primary goal of this approach is to restore end-organ perfusion, without addressing the pathologic aortic problem directly (ie, repair of primary tear or prevention of false lumen expansion). Although the focus of this report is type B dissections, we have reported this as an effective means to restore end-organ perfusion in type A dissection presenting with malperfusion.

Thoracic endovascular repair (TEVAR) has been increasingly utilized as an alternative to percutaneous fenestration. With this approach, the endograft is deployed to cover the primary entry tear within the true lumen. As a result, the true lumen then expands, and dynamic branch vessel compromise is ameliorated. By eliminating flow through the primary entry tear, false lumen flow is significantly reduced, and its thrombosis then induced. If then present, static branch vessel obstruction is treated with self-expanding stents as above. In contrast to percutaneous fenestration and true lumen stenting, this approach may treat both the malperfusion component as well as the primary pathologic aortic problem.

We find that preoperative workup for either approach includes the need for dynamic computed tomography (CT) scanning from the thoracic inlet to the femoral heads. Key information gleaned from this examination includes identifying whether and by what mechanism branch vessel obstruction exists, and whether an associated aortic aneurysm is present. The latter is an important determinant of proceeding with TEVAR as opposed to percutaneous fenestration. We have noted during the intervention, however, that multiple branch vessels may be compromised, when in fact only one vascular bed was suggested as being affected on preoperative clinical or imaging examination.

There cannot be enough emphasis placed on attention to preoperative endograft planning, as the durability of TEVAR depends not only on stability of the proximal landing zone but also on the accuracy of the sizing. We have found that 3D reconstructions of CT scans are quite useful both in determining the suitability of TEVAR as well as in assisting with sizing considerations. This adjunct also aids in determining the access vessels in TEVAR. For TEVAR (in contrast to per-

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cutaneous fenestration), we have had a liberal policy toward using a lumbar drain to reduce intrathecal pressure and potentially reduce the dreaded postoperative complication of paraplegia. These are typically placed before the procedure and left in place for 36 to 48 hours postoperatively. Permissive hypertension (keeping the spinal perfusion pressure over 80 mm Hg) is also used as an adjunct for the first 48 to 72 hours.

Operative Techniques

Thoracic Aortic Endovascular Repair

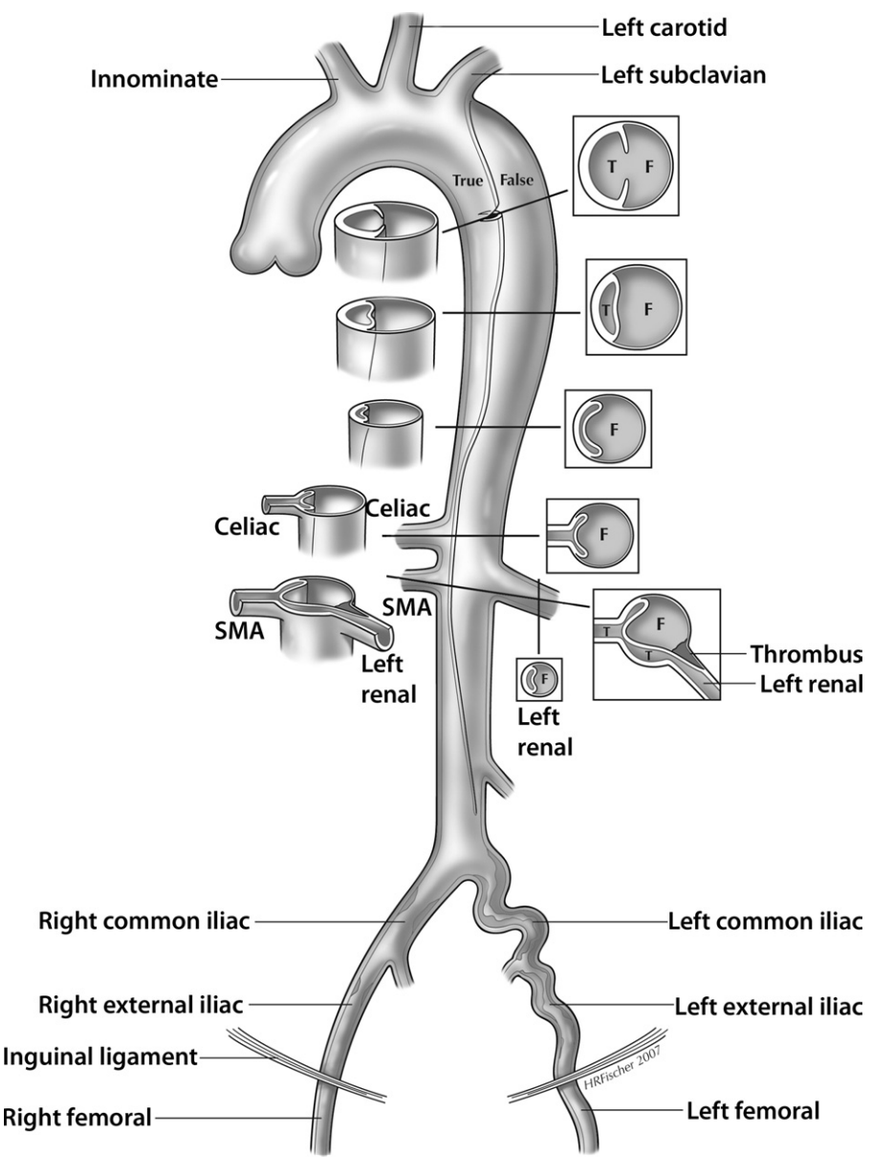

Figure 1 A type B aortic dissection is depicted detailing the anatomy. Intravascular ultrasonography (IVUS, catheter not shown) is an invaluable aid in treatment of B-AD with malperfusion. The cross-sectional images of the aorta and branch vessels correlate to the images seen on IVUS. By depicting the relation of the dissection flap to branch artery origins, IVUS demonstrates which branches are likely to be compromised and by what mechanism. Because of the high frequency of multi-organ compromise, branch artery manometry and arteriography with hand-injections of contrast (not shown) are performed to confirm patency and perfusion of renal, superior mesenteric, and if indicated, the iliac arteries. In this figure, the primary entry tear is situated in the proximal descending thoracic aorta. The dissection flap distal to this shows evidence of collapse of the true lumen with dynamic obstruction of the celiac and superior mesenteric arteries. In these vessels, the dissection does not extend into them but rather occludes them by intermittent obstruction of the flap during the cardiac cycle. In contrast, the left renal artery shows evidence of dissection without reentry in the course of the branch vessel. In this branch, there is formation of thrombus in the left renal artery false lumen, which in turn causes a static obstruction and renal malperfusion. F = false lumen; SMA = superior mesenteric artery; T = true lumen.

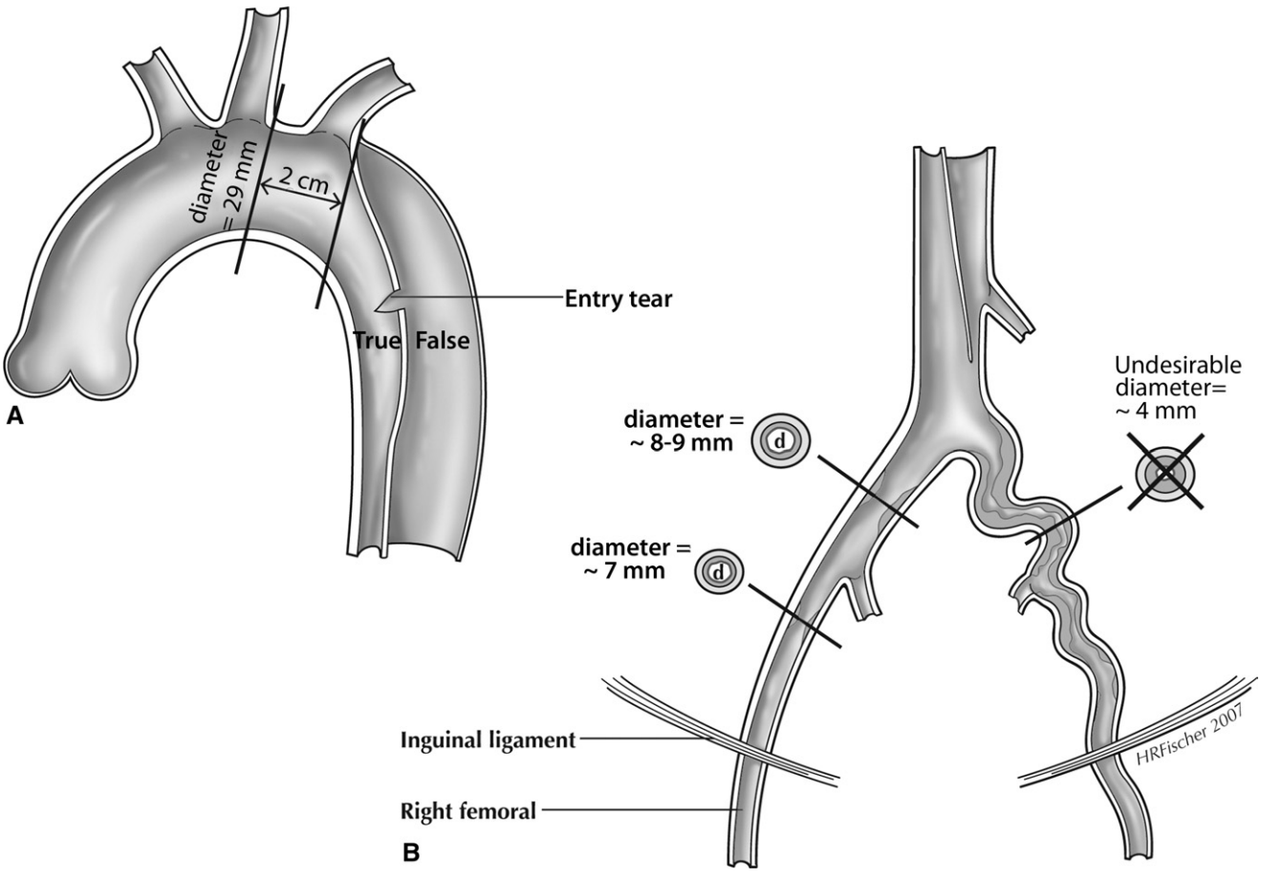

Figure 2 Preoperative CT scanning is essential to determine adequacy of the proximal landing zone. We typically extend coverage up to the left subclavian artery in cases of type B aortic dissection, often noting the need for complete coverage to obtain a 2-cm landing zone (A). The only commercially available endograft comes in sizes from 26 to 40 mm in diameter. In determining the correct endograft size, we obtain measurements at the proximal edge of the pathologic problem and then at 1-cm proximal increments. The selected endograft then has a diameter no more than 10% larger than the aortic diameter at the landing zone. For (A), a 31-mm graft would be selected. Finally a short (10-15 cm) stent graft length is commonly utilized in our experience to avoid extensive intercostal artery coverage. Additional anatomic requirements for TEVAR include the absence of a tapered neck, the presence of a 2-cm proximal margin, and a relatively "flat" arch (in contrast to a "Gothic" arch) to allow for suitable apposition of the endograft to the aortic lesser curvature. Finally, the access vessels are determined. The ideal vessel is straight, not calcified, and of adequate diameter to accommodate the delivery sheath for the endograft (ie, the right femoral artery in B). We have liberally used intraoperative iliac angioplasty to allow for femoral delivery in cases of short discrete iliac stenosis. If these access vessel requirements are not met, we plan delivery via a conduit placed on the common iliac artery. In our experience, the iliac arteries of patients with aortic dissection are more often ectatic than stenotic, and the need for adjunctive iliac angioplasty is reduced compared with the population of patients with degenerative thoracic aneurysms. d = luminal diameter.

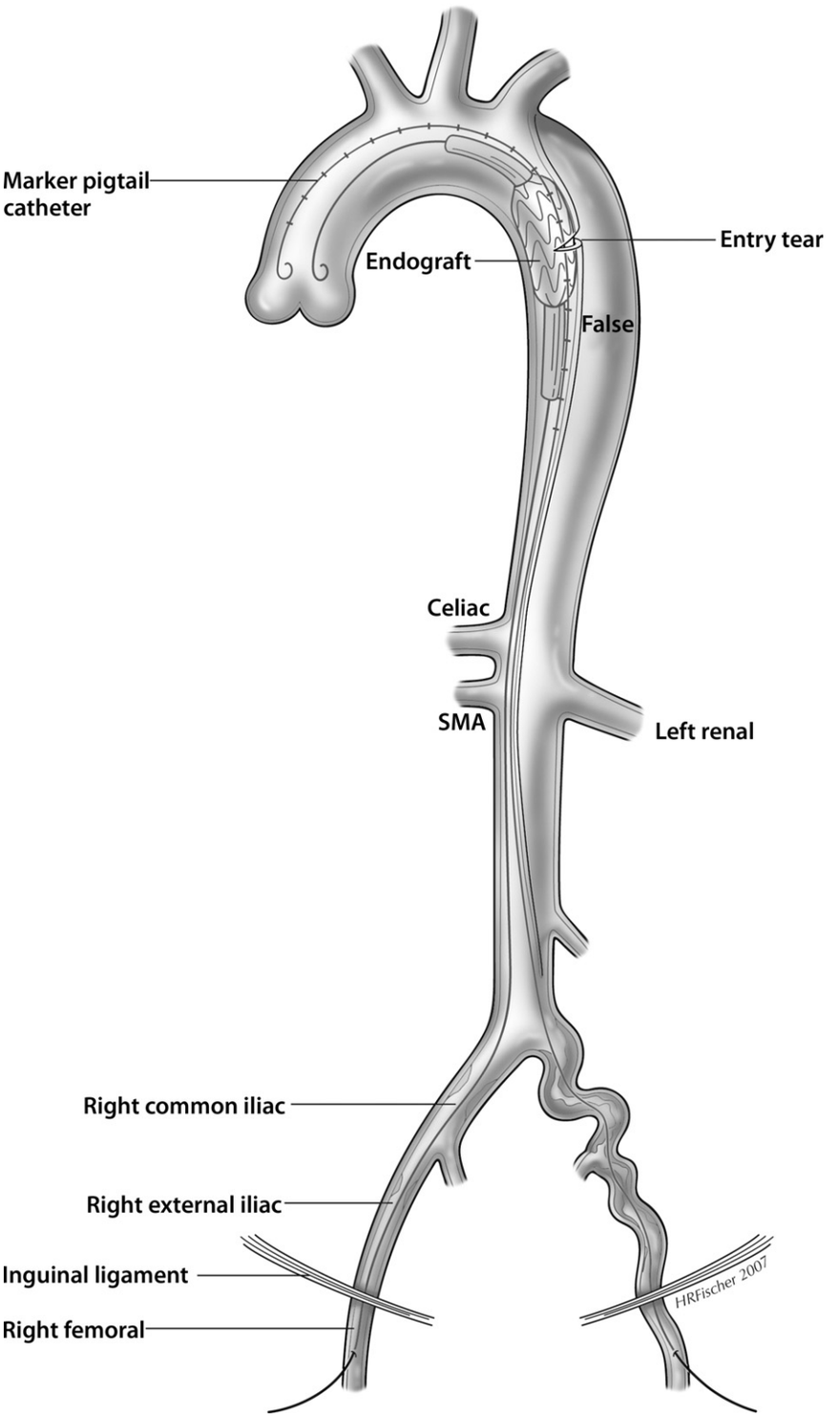

Figure 3 After IVUS examination as well as initial angiography of the arch to determine great vessel origin (Fig. 1), the access vessels are cannulated. The tortuous left iliac artery in this case serves as the route for percutaneous placement of a marker pigtail catheter, used for both angiography and assistance in marking the site of deployment. Via an open exposure of the right femoral artery, the delivery sheath is placed into the terminal aorta (not shown) over a stiff Lunderquist wire. The wire position is maintained typically in the ascending aorta throughout the procedure. Note that it is important to ensure true lumen placement of both wires to avoid the catastrophic complications of false lumen endograft deployment or of extension of dissection to a type A. Finally, the endograft is situated in the correct site and deployment is begun. SMA = superior mesenteric artery.

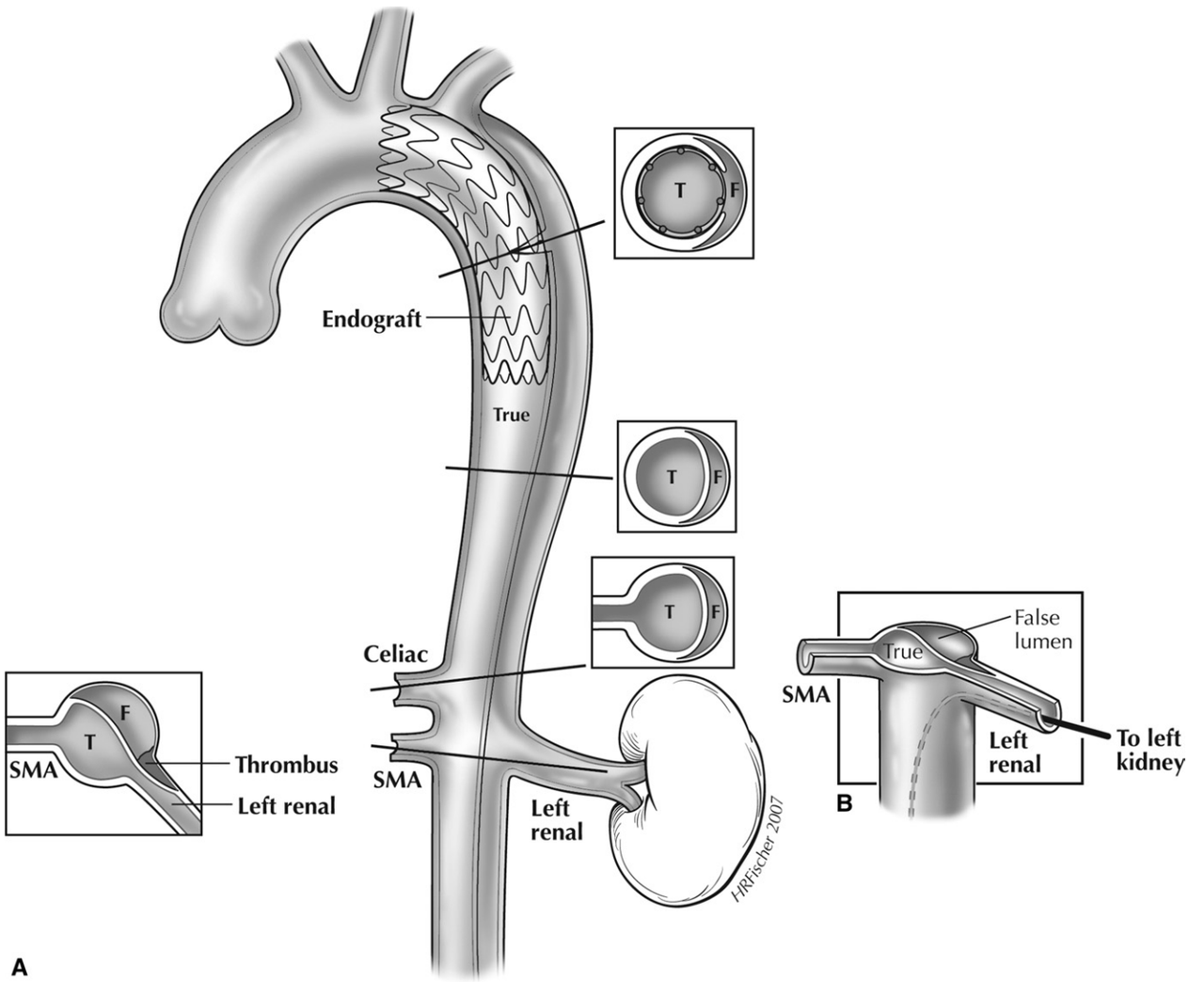

Figure 4 After endograft deployment, balloon dilation to profile only can be performed at the proximal landing zone only (not shown). We have been somewhat reluctant to do this for fear of tearing the already inflamed aorta. Completion aortography demonstrates accurate deployment and elimination of flow via the entry tear. IVUS examination of the remaining nontreated aorta confirms resolution of dynamic obstruction (A). However, as is shown in this figure, the left renal artery demonstrates static obstruction and this is confirmed by obtaining pressure gradients from the aorta to the renal hilum in the renal artery true lumen (B). F = false lumen; SMA = superior mesenteric artery; T = true lumen.

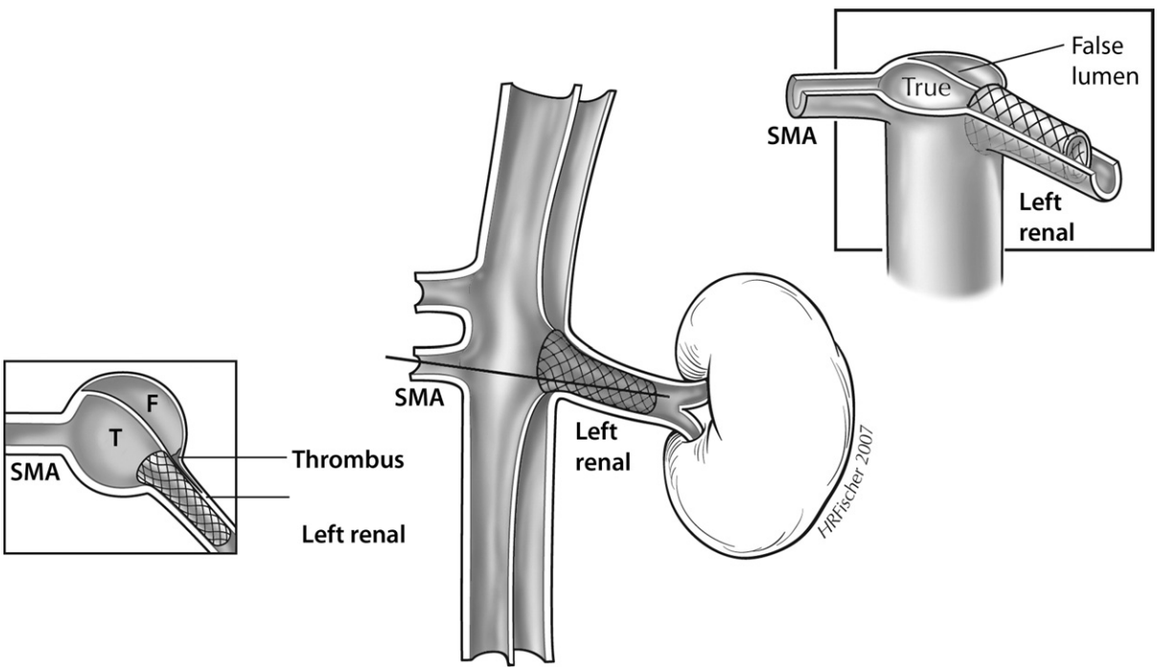

Figure 5 In the event of angiographically significant stenosis, or a measurement of a 20 mm Hg systolic gradient, the renal true lumen is stented with a self-expanding stent that is sized 10% more than the diameter of the renal artery. Completion manometry demonstrates branch vessel patency and adequate antegrade branch vessel flow. F = false lumen; SMA = superior mesenteric artery; T = true lumen.

Percutaneous Fenestration and Stenting

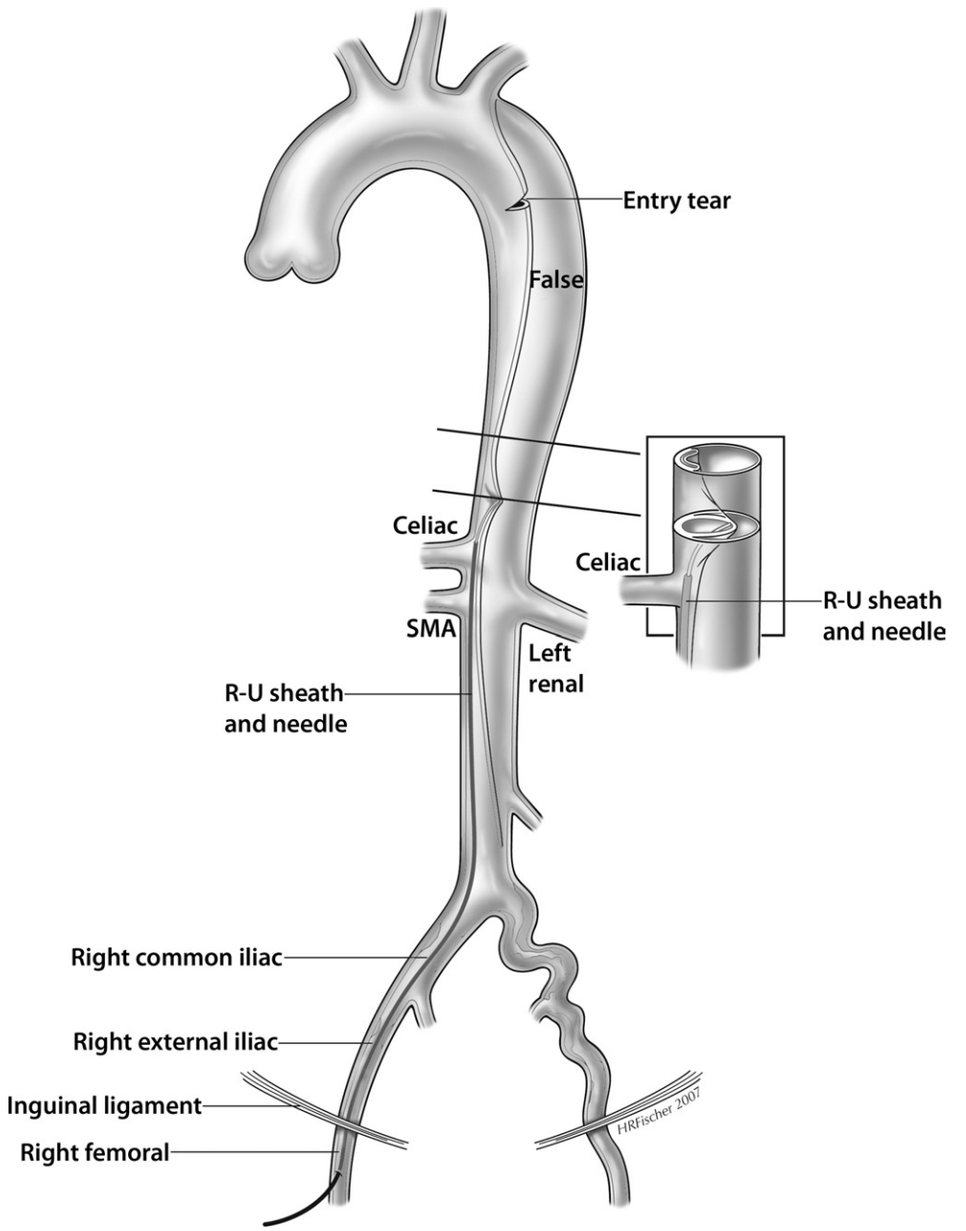

Figure 6 This figure again depicts the aorta after angiography and IVUS examination. Access for this procedure is entirely percutaneous. The entry tear site is confirmed, and there is evidence of dynamic and static branch vessel flow. Pressure measurements are obtained at the aortic root, and in selected branch vessels (not shown). When branch vessel obstruction is present, the goal is to reperfuse the mesenteric vessels first, followed by renal and limb revascularization. R-U = Rosch-Uchida needle; SMA = superior mesenteric artery.

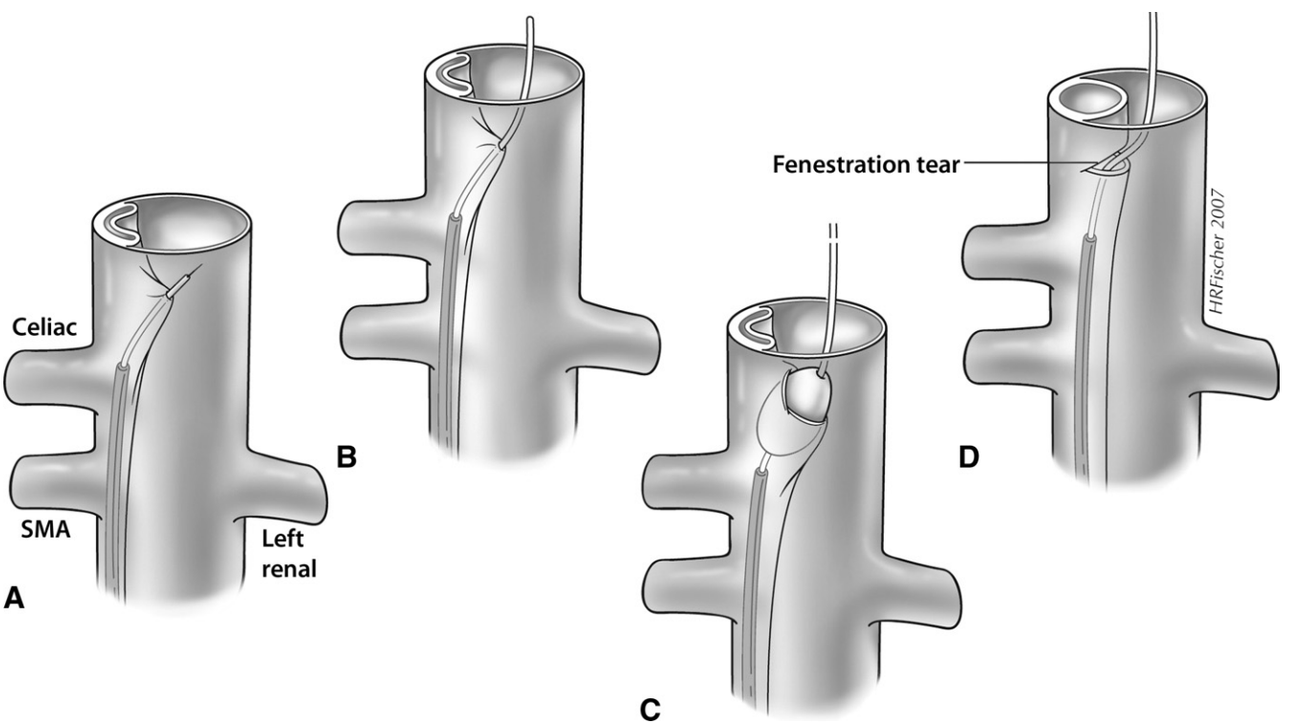

Figure 7 The true lumen is cannulated and a Rosch-Uchida needle (Cook, Bloomington, IN) is then placed from the true into the false lumen in a perpendicular manner with IVUS monitoring (A). The site of fenestration is at the branch vessels that are intended for therapy. A wire and subsequently a 5-Fr catheter are then placed into the false lumen (B). IVUS or small-dose contrast delivery can confirm the false lumen location of the catheter. A 14-mm balloon is then placed at the puncture hole in the flap (C) and then inflated often without seeing a "waist" to create a fenestration (D). RU = Rosch-Uchida needle; SMA = superior mesenteric artery.

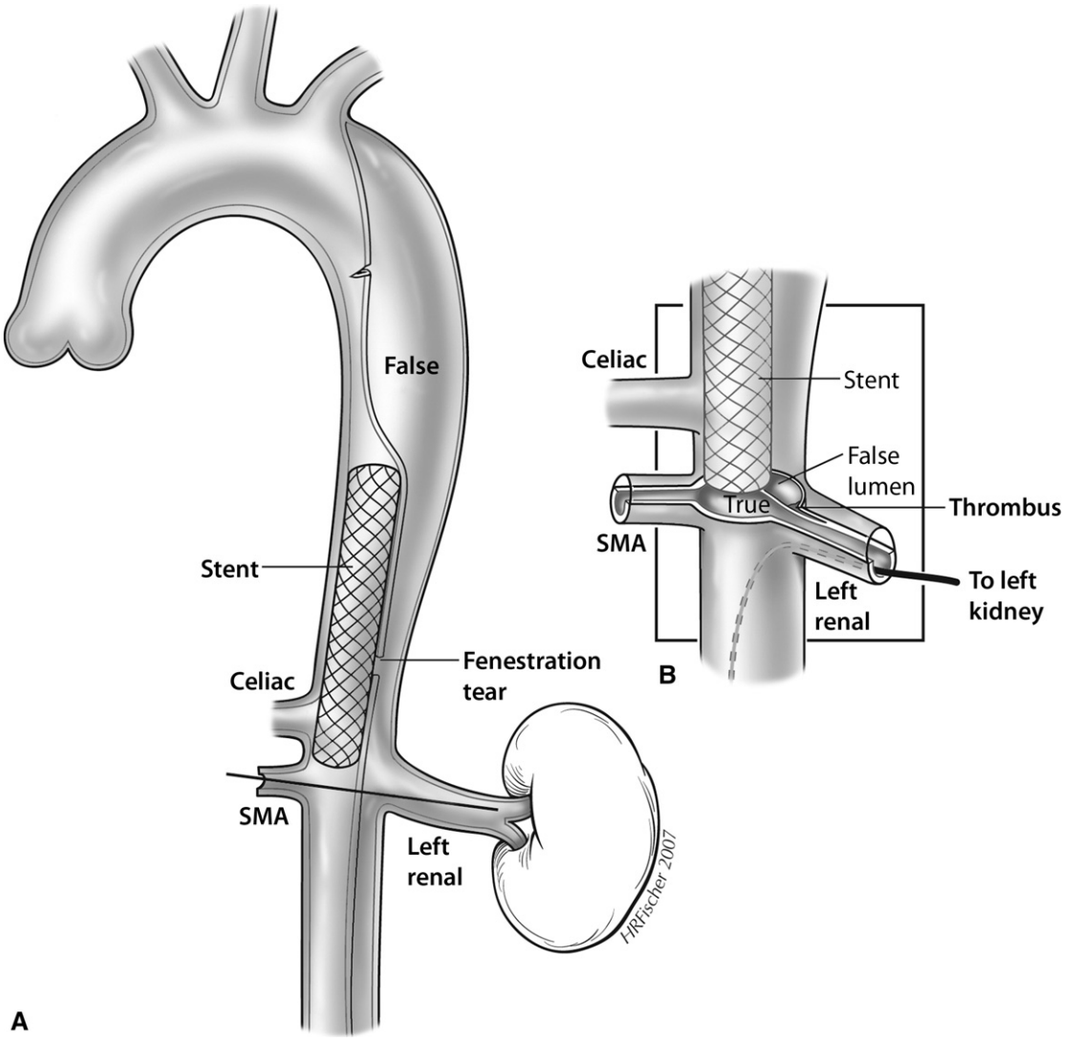

Figure 8 Although fenestration can equalize pressures across the dissection flap, self-expanding 16- to 22-mm-diameter Wallstents (Schneider, Minneapolis, MN) are usually placed to buttress open the aortic true lumen and are typically placed near the compromised branch vessel (A). These are usually inserted via the already obtained percutaneous access sheaths and deployed by IVUS guidance. Care is taken to withdraw the guidewire from across the fenestration tear and re-advance it within the aortic true lumen, so as to deploy the stents exclusively within the aortic true lumen, rather than straddle the tear from true to false lumen. Neglecting this critical step adds greatly to the complexity of the procedure. Fenestration and stenting of the aortic true lumen treats the dynamic obstruction of the depicted mesenteric vessels, but not the static obstruction seen in the depicted left renal artery. In this branch, pressure measurements confirm a significant (≥20 mm Hg) systolic gradient requiring additional treatment (B). SMA = superior mesenteric artery.

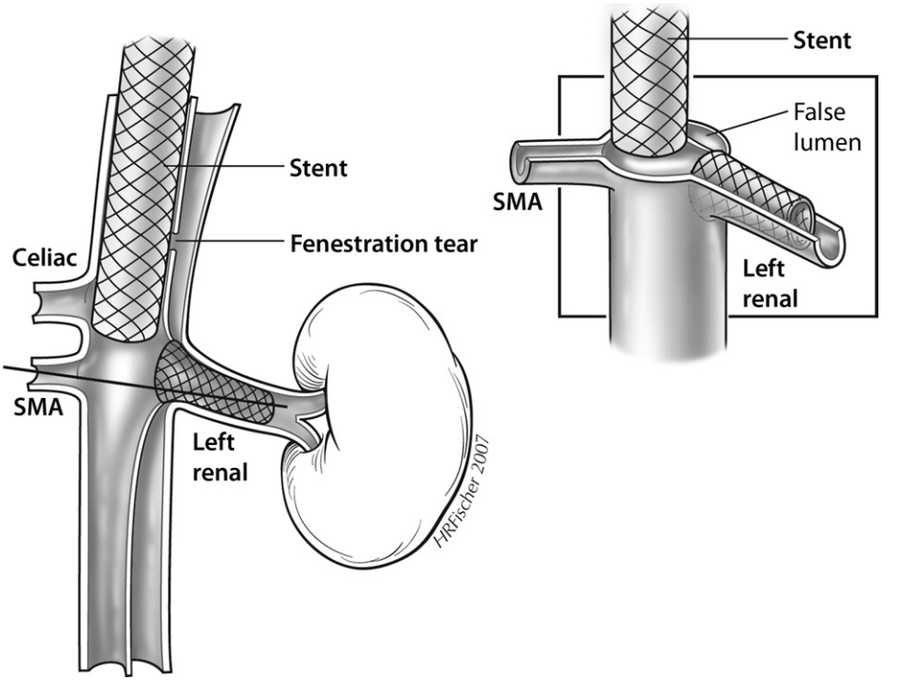

Figure 9 Again, the true lumen of the left renal artery that is compromised by a static obstruction is cannulated and treated with a self-expanding stent to relieve the obstruction. Completion aortorenal manometry confirms adequate perfusion. SMA = superior mesenteric artery.

Conclusions

At the University of Michigan Hospital, we have evaluated 205 patients for either type A or B dissection with malperfusion from 1997 to 2007. An additional 33 patients have undergone TEVAR for treatment of acute (true "double-barrel" dissection or its variants of intramural hematoma with or without penetrating ulcer) or chronic dissection since the inception of our thoracic endovascular program in 1993.[3,4] Although the long-term results of percutaneous fenestration and stenting are documented, the results from TEVAR in this setting remain to be defined. We have found that TEVAR in patients with acute dissection variants of intramural hematoma with or without penetrating ulcer to be particularly vexing with a high incidence of fixation site complications (eg, pseudoaneurysm formation).[3] With recognition of these problems, we have evolved our technique to avoid ballooning the endograft after deployment particularly in areas of dissected aorta. When the dissection extends proximally to near the subclavian artery, we try to land the stent graft proximally to cover the left subclavian artery. We do this partly because we have noted from open surgery that even though this area may not have a true dissection, it is still quite inflamed and may potentially serve as a poor proximal fixation site. Finally, we believe that, in the setting of infradiaphragmatic malperfusion, the benchmark for TEVAR remains percutaneous fenestration and stenting with subsequent elective open repair if needed for aneurysmal degeneration, given its known documented durable results.

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